UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF NEW YORK

James H. Sarkees and Deborah J. Sarkees,

Plaintiffs,

v.

E. I. DuPont de Nemours and Company *et al.*,

Defendants.

Report and Recommendation

17-CV-651 (JLS)

I. INTRODUCTION

In May 1974, a 19-year-old James Sarkees started a job at the Goodyear Tire & Rubber Company in Niagara Falls, New York ("Goodyear"). James took the job as "summer help," which meant, among other things, that he was a non-union worker in a union shop. (*See* Dkt. No. 56-6 at 9; Dkt. No. 56-7 at 2; Dkt. No. 70-5 at 58.) From May 1974 until he left Goodyear in December 1974, James worked jobs at Goodyear that gave him low, moderate, or high-level exposure to an aromatic amine called *ortho*-toluidine ("OT"). OT has been investigated for decades as a suspected human carcinogen. After leaving Goodyear, James worked a few years at the Dunlop Tire factory in Tonawanda, New York and had a stint at a Union Carbide facility. James then spent the rest of his career working various jobs in the trucking industry.

In 2016, at the age of 61, James was diagnosed with bladder cancer. The diagnosis occurred during regular screening that James received for years through a monitoring program for former Goodyear workers. Goodyear implemented the monitoring because researchers and experts in the chemical industry knew for decades that OT, at a minimum, had a suspicious association with increased rates of bladder cancer. Given that bladder cancer often has a long latency period, James

and his wife Deborah began to suspect that exposure to OT at Goodyear was a substantial factor in causing his bladder cancer.

Acting on their suspicions, James and Deborah sued defendants E. I. DuPont de Nemours and Company and First Chemical Corporation for negligence, strict product liability, and loss of consortium. Plaintiffs also sought punitive damages. Following discovery, defendants filed three motions that currently are pending. (Dkt. Nos. 56, 59.) Defendants seek summary judgment on all of plaintiffs' claims under Rule 56 of the Federal Rules of Civil Procedure. Defendants also seek to preclude the testimony of plaintiffs' expert on general causation, Dr. Ronald L. Melnick, and their expert on specific causation, Dr. Christine Oliver. The motions contain a lot of technical information that the Court will explain more below. In short, defendants believe that summary judgment and expert preclusion are appropriate because the research literature on OT relies too much on animal studies that do not have direct human implications and epidemiological studies that do not address the duration of exposure that James had at Goodyear. Defendants also assert that plaintiffs have not made nearly enough of a showing to go to trial on punitive damages and that Deborah's claim for consortium must fail because it arose after James's alleged exposure. Plaintiffs' argue that all of their claims should go to trial in large part because numerous studies over many decades have generated reliable information about how OT probably acts as a mutagen and about how OT has epidemiological associations with elevated risks for bladder cancer.

This case was referred to this Court under 28 U.S.C. § 636(b).[1] (Dkt. No. 9.) The Court has deemed the motions submitted on papers under Rule 78(b). For the reasons below, the Court

[1] District Judge Lawrence Vilardo made the referral and originally presided over the case. The case since has been reassigned to newly appointed District Judge John L. Sinatra, Jr. (Dkt. No. 76.)

respectfully recommends denying defendants' motions except to grant defendants summary judgment for Deborah's claim for loss of consortium.

II. BACKGROUND

A. James's Work at Goodyear

This case concerns allegations that defendants harmed James by failing to warn him or Goodyear of the risk of developing bladder cancer from exposure to OT. James began working for Goodyear on May 30, 1974. James's first job with Goodyear was as a production operator in a department called Department 245. James worked as a production operator until August 30, 1974 and then again from September 4 to October 12, 1974. On October 12, 1974, James started work as a lab technician in Department 460. James ended his time with Goodyear on December 29, 1974. (Dkt. No. 56-7 at 2.) James worked other jobs over the course of his career before retiring. In 2016, James was diagnosed with bladder cancer.

James's exposure to OT while at Goodyear is central to this litigation. While a production operator, James would work a cycle of seven consecutive days with two days off and could wind up working any of three shifts. Job responsibilities included bagging the chemicals Nailax™ (also branded as Wing-Stay™) and Kagarax™. Nailax was an antioxidant that preserved the integrity of the rubber used during tire production. Nailax contained OT, aniline, hydroquinone, and toluene. (Dkt. No. 56-8 at 8.) "Orthotoluidine is used in the rubber industry as an antioxidant, as a rubber vulcanization accelerator, and to protect tire cord against heat aging." (Dkt. No. 57-3 at 3.) Kagarax also was an accelerator and contained aniline, nitrobenzene, hydrogen sulfide, and carbon disulfide. (Dkt. No. 56-8 at 8.) James occasionally loaded and stacked pallets with Kagarax. James also cleaned the two chemical reactors in Department 245 as well as the sparkler filter. James worked at the tank farm in Department 245 as well, unloading tank cars. James's work attire consisted of steel-

toed work boots, jeans, a T-shirt, and safety glasses. (Dkt. No. 56-6 at 36.) As a lab technician, James frequently tested the specific gravity of product samples that came from the tank farm in Department 245. The product samples covered a variety of different chemicals and were not limited to any chemicals containing OT.

In her report, Dr. Oliver provided extensive factual detail for the various tasks that James performed while at Goodyear. Since the core essentials of James's work—aside from the scientific and legal significance of some details, which will be addressed later—are not disputed, the Court will quote those facts from the report for background. (*See also* Dkt. Nos. 56-2 at 5, 56-14, 70-6 at 49–53.) Dr. Oliver described James's bagging responsibilities as follows:

> The Nailax production process is outlined in referenced reports of findings in March and October of 1979. Two Nailax reactors were located on the upper level of Building 32. Hydroquinone, orthotoluidine, and aniline were added to the reactor vessels, along with toluene and an iron catalyst. Opportunities for exposure to ortho-toluidine and aniline existed at the weight scales for each; neither had automatic shut-off controls to prevent accidental overflow. Incomplete reactions also provided opportunity for accidental exposure. Unreacted materials were removed from the reactor degasser and from there to recycle tanks. Spills from the recycle tank resulted in excessive exposure from dermal contact. As a production operator, Mr. Sarkees did not work in proximity to the recycle tank.
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> Molten Nailax was piped to the ground level where it was filtered to remove neutralized catalyst. The primary filter had to be cleaned after two batches and the secondary filter, after 10 batches. The primary filter was changed as often as five times per shift. (According to Mr. Sarkees, at the time he worked at Goodyear, there was only one filter.) The changing/cleaning of the filters were done manually and workers exposed to vapors from the molten material. At the time of the industrial hygiene survey the canopy exhaust hood over the filters was ineffective.
>
> Molten Nailax was piped from holding tanks to a flaker that cooled, solidified, and flaked the material. It was then put on a conveyer belt and transferred Building 33 where it was bagged.
>
> Answers to Interrogatories, his deposition testimony, and my interview indicate that Mr. Sarkees performed five tasks that involved work with and exposure to ortho-toluidine during the Nailax production process. These were cleaning the two Nailax reactors, cleaning the Sparkler Filters, unloading the tank cars in the Tank Farm, bagging and drumming Nailax, and driving the forklift in the Shipping

Department for five to seven days. Mr. Sarkees testified at his deposition that at each of the first three tasks he came close to being overcome with fumes at times.

(Dkt. No. 56-8 at 8–9.) Dr. Oliver described James's reactor cleaning as follows:

> In his Answers to Interrogatories Mr. Sarkees testified that each of the two reactor vessels were shut down for one to two days a month for cleaning and repairs. Cleaning required him to enter the vessel from the second floor mezzanine through a hatch 2 feet in diameter. An electric work light and an air ventilation hose were lowered into the vessel. Respiratory protection was supplied in the form of a half-face cartridge respirator. He was not fit-tested for use of the respirator. He wore paper coveralls and black vinyl gloves which were taped to the coveralls. He also wore a safety harness with a rope attached in case he was overcome by fumes.
>
> Prior to cleaning, the reactor vessels were allowed to cool down. Mr. Sarkees climbed down a ladder into the reactor vessel. There he used a hammer and chisel to remove solidified material from the walls of the vessel, the mixing shaft, the flanges, and the fittings. Other workers lowered buckets into the vessel to receive the material that had been removed. He testified at his deposition that when shoveling out the liquid/solid Nailax residue at the bottom of the reactor he splashed some on his clothing and occasionally on his skin (p. 165).
>
> Mr. Sarkees testified that it was hot and humid in the vessel, so that his safety glasses and respirator were poorly adherent to his face. He had to remove his respirator periodically in order to breathe.
>
> Each operator worked in turn inside the reactor vessel for about 30 minutes before being pulled out to get fresh air. This process went on until the supervisor determined that the reactor was clean enough. Mr. Sarkees estimated that he spent 40 hours cleaning the reactors while he worked in Department 245 at Goodyear—10 cleanings at 4 hours each. In addition, he wore the same contaminated coveralls for the entire work shift. He reported that because the hatch into the reactor vessel was about two feet in diameter and because he was young and "skinny", he was more likely to be assigned to clean the Nailax reactor during the months that he worked at Goodyear.

(*Id.* at 9.) Dr. Oliver described James's cleaning of sparkler filters as follows:

> Mr. Sarkees estimated that he cleaned the Sparkler Filters more than 80 times, with each cleaning taking about 1.5 hours. Two men were required. He wore safety glasses, steel-toed work boots, a T-shirt or a long-sleeved work shirt, jeans, and black vinyl gloves. He did not wear respiratory protection.
>
> The first step was to remove the top of the filter housing. He testified that "It was hot, smelly, and the fumes would choke you." The next step was removal of the filter assembly from the housing using an overhead electric lift. The assembly

was 3 ft wide and 4.5 ft. tall. It had a strong chemical smell, with fumes coming out of every layer, according to Mr. Sarkees' description. Dark brown filter cake containing both liquid and solid material coated the layers. Rods ran down the outside of the filter assembly. These were removed, leaving 15 to 18 round steel screen trays accessible. Between each tray was filter paper. The two operators would pick up each tray by handles welded to the side and dump the dirty caked filter paper into open 55-gallon drums or small dumpster bins. The filter paper was hot and releasing fume[s]. After unstacking and cleaning the dirty filter assembly, the men would reassemble a clean filter. That filter was reloaded into the filter assembly housing.

According to his report to me, he cleaned the Sparkler filter twice a day on average. Exposure to Nailax was by inhalation and dermal contact.

(*Id.* at 10.) Dr. Oliver described James's unloading duties as follows:

For the first week of his employment, Mr. Sarkees relieved other workers who were unloading tank cars. He monitored storage tank levels and watched for leaks or malfunctions. He was then shown how to unload the tank cars himself. After checking paperwork with the supervisor, he would check that the wheels were blocked and the tank grounded. An air valve was opened from inside the tank car, releasing pressure and fumes. He testified at his deposition that he climbed to the top of the tank car, opened the hatch, and took samples. The samples were analyzed in the lab and he let the supervisor know the results. If the testing was acceptable, he took a hose over to the tank car and attached it to "the bottom fidget" on the tank car (p. 179, line 23), opened a manual valve, turned on a pump inside, and began unloading the chemical to a storage tank in the tank farm.

In his Answers to Interrogatories Mr. Sarkees estimated that he oversaw sampling and unloading of 35 or more railroad tank cars; approximately 20 contained ortho-toluidine.[2] He recognized both ortho-toluidine and aniline by the smell. He noted that "During the summer, the fumes of the ortho-toluidine would come out of the hatch of the tank car and would sometimes take my breath away and choke me." At his deposition, Mr. Sarkees testified that he spent about 75% of his time actually taking samples and unloading the cars and 25% overseeing the sampling process (p. 197, lines 8-11). At this job he wore safety glasses, T-shirt or long-sleeved work shirt, jeans, and work boots. He was not provided with a respirator.

Once the car was emptied, Mr. Sarkees closed the valve at the bottom, shut off the pump, closed the valves in the pump house, flushed out the hose with water, checked the level in the tank car and closed the top, and gave his supervisor a reading. He testified at his deposition that he could smell the fumes from the tank

[2] There is some information in the record suggesting that the tank cars had a capacity of 10,000 gallons each and that OT weighed about 8.3 pounds per gallon, meaning that a tank car full of OT would have contained about 83,000 pounds of the chemical. (Dkt. No. 70-8 at 67.)

car but was never overcome. Chemicals spilled and splashed on his skin in the sampling process (p. 199). In the pump house, chemicals were leaking onto and collecting on the floor. He was exposed to vapors from these chemicals when he was in the pump house.

(*Id.* at 10.) Dr. Oliver provided further details about James's bagging and drumming responsibilities:

> At this job, he wore cotton gloves or no gloves at all. (He did not recall the frequency of his use of gloves.) Otherwise his protective clothing was as described for the other jobs. He was not given respiratory protection. At his deposition, Mr. Sarkees described the bagging and drumming process as follows (pp. 149, 150): The product came out of the conveyor belt. If "you had the first position", you took a bag out of the box, opened it up, put it on a scale and filled it with the material on the small feed conveyor. When the weight reached 50 lbs., it was sent on to the next person. That person sewed the top closed and moved it from the belt to a pallet. Bags were layered on the pallet, five to a layer, with glue added to the top to keep them from slipping. Forty to fifty bags were put on a pallet. Open-top fiber drums were filled in a similar fashion and put on pallets.
>
> History obtained from Mr. Sarkees at the time of my interview reveals that there was an open vat of liquid Nailax behind those bagging and drumming, a vat that released chemical fumes into the air. A large roller picked up the liquid Nailax and spread it onto a belt for cooling and flaking. One of the two workers on the bagging line had to stir the bottom of the vat periodically to keep it liquid. The workers rotated stirring. There was exposure to vapors from the open vat during bagging and during the stirring of the liquid.
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> In his Answers to Interrogatories, Mr. Sarkees stated that the work area was dirty and dusty. He cleaned dust off his clothes and skin with an air hose. His eyes were irritated and itchy. Mr. Sarkees estimated that he did this job for 1.5 to 2 hours each day, sometimes covering other workers on break and at lunch.

(*Id.* at 11.) Dr. Oliver summarized what James did as a forklift driver:

> For a five- to- seven- day period Mr. Sarkees filled in for an employee in the Shipping Department. His job was to locate product and load it onto a trailer—either at a dock or attached to a tractor. He blew dust off the pallets of product and the dust became airborne in his breathing zone. He had no respiratory protection. The products were Nailax and Kagarax.

(*Id.*) Finally, Dr. Oliver summarized what James did as a lab technician:

> From October 12 to December 29, 1974 Mr. Sarkees worked as a laboratory technician in Department 460. According to his deposition testimony and his

> Answers to Interrogatories, his job was to run analytical tests on by-product material from the Nailax reactors and samples of raw material brought directly from the tank farm. Samples were poured from plastic sample bottles into a graduated cylinder. Liquid was present on the outside of the sample bottles and got on his hands when he poured from the sample bottles. There was opportunity for dermal contact when he poured the samples back into the sample bottles.
>
> Mr. Sarkees estimated that he spent about four hours each work day during this period testing raw materials, by-product from Department 245, and samples from the tank farm. Testing on ortho-toluidine specifically was performed by him about 15 times by his estimate, taking about 20 minutes each time.
>
> He did not recall wearing gloves and he did not have respiratory protection, and at his deposition he did not recall a fume hood (p. 212).

(*Id.*)

B. Work After Goodyear

From 1976 to 1980, James worked at the Dunlop Tire factory in Tonawanda, New York. During his time at Dunlop Tire, James worked as a laboratory technician and a process control technician. James's work areas were clean, and he was not exposed to any chemicals or fumes. From 1980 to 2015, James worked as a truck driver and briefly as a worker at a Union Carbide facility. Other than incidental outdoor exposure to some diesel fuel vapor when refueling, James's trucking assignments over the years did not expose him to chemicals.

C. Diagnosis

In 1998, James entered the bladder cancer screening program that Goodyear offered for people who worked at its factory. As early as 2003, James tested positive for "atypical cells," though follow-up cystoscopy found no evidence of bladder cancer. In 2012, routine screening found atypical cells again; this finding was monitored for the next few years. In 2016, doctors uncovered a lesion that led to follow-up testing including a biopsy. James was diagnosed with bladder cancer in November 2016. On December 19, 2016, a physician at the health provider conducting the Goodyear-sponsored screening wrote a letter ruling out other factors that could have elevated

James's risk for bladder cancer. The physician concluded that James's bladder cancer was "directly related to his exposure while employed at Goodyear." (Dkt. No. 56-8 at 6 (quoted in Dr. Oliver's report).)

D. Science of OT Exposure / Review of the Literature

As far back as 1895, researchers began to notice a curious association between aromatic amines—the family of benzene-ring chemicals that includes OT (*see* Dkt. No. 57-1 at 4 ("o-Toluidine is a toxic aromatic amine"))—and increased occurrences of bladder cancer. (Dkt. No. 56-28 at 2.) Investigations of the association continued into the 1970s. (*Id.*) In December 1989, the National Institute for Occupational Safety and Health ("NIOSH") issued an interim report of its health hazard evaluation of the Goodyear plant. (Dkt. No. 56-14.) The labor union at the plant requested the evaluation because eight cases of bladder cancer were reported between 1973 and 1988 among current and former employees. Out of 1,749 people ever employed at the plant in any capacity, 14 cases of bladder cancer were observed when statistical and epidemiological analysis suggested that 3.54 cases should have been observed. Within the overall population, 795 people worked in Department 245, where James worked; of these people, eight cases were observed when 1.2 cases would have been expected. The interim report concluded that "the excess risk of bladder cancer is associated with exposure to OT and aniline." (*Id.* at 3.) The interim report discussed studies and data produced over the years by the International Agency for Research on Cancer ("IARC") and the National Cancer Institute ("NCI"). Based on a review of carcinogenic data in the 1980s, the IARC concluded that, while OT could not be identified specifically as the cause of bladder cancer in workers exposed to other chemicals at the same time, "[OT] should be regarded, for practical purposes, as if it presented a carcinogenic risk to humans." (Dkt. No. 56-14 at 7.) NCI and other agencies published reports of animal studies involving OT. Those animal studies

indicated an occurrence of multiple types of cancers in rats at lower doses than aniline. (*Id.*) NIOSH described how its assessment of the Goodyear plant included air sampling in 1988. NIOSH also described different recommended levels by different government agencies. In short, other government agencies have classified OT as a suspect carcinogen and have recommended that occupational exposure by error not exceed either five parts per million (5 ppm) or two parts per million (2 ppm). (*Id.* at 10–11.) NIOSH had no recommended exposure limit for OT. (*Id.*) NIOSH, having evaluated the plant in 1988, could not quantify levels of exposure to OT for years prior to 1982. (*Id.* at 13.) Nonetheless, NIOSH was able to show that "[t]he risk of bladder cancer is statistically increased in the cohort as a whole, but the highest risk is observed among workers who had ever been employed in Department 245. Workers in this Department had an SIR [standardized risk ratio] of 6.64, which implies that they were 6.64 times more likely than New York State residents of similar age and sex to develop bladder cancer." (*Id.* at 14.) At the end of the interim report, NIOSH discussed how

> There are several methodological reasons why our study would tend to underestimate the risk of bladder cancer. Firstly, we may have missed cases that occurred among former workers who have moved out of New York State. Currently, about 28 percent of cohort members have addresses outside New York State. Secondly, we assumed that all cohort members not known to have died or developed bladder cancer were alive and free of disease until the end of the study.

(*Id.* at 16.)

In December 1990, NIOSH issued an alert titled, "Request for Assistance in Preventing Bladder Cancer from Exposure to [OT] and Aniline." (Dkt. No. 56-15.) "This Alert presents significant new epidemiological evidence that clearly associates [OT] and aniline with an increased risk of bladder cancer." (*Id.* at 4.) NIOSH cited multiple animal studies from the 1970s showing how OT induced a variety of cancers in rats and mice. (*Id.* at 5.) NIOSH also reviewed limited human studies; while these studies had other chemicals as confounding variables, these studies noted

62-fold and 72-fold increases of bladder cancer among factory workers exposed to chemicals including OT. (*Id.* at 6.) NIOSH ended the alert by discussing the Goodyear plant.

In 2010, lead authors Tania Carreón, Misty J. Hein, and Susan M. Viet published a study titled, "*Increased bladder cancer risk among workers exposed to* o*-toluidine and aniline: a reanalysis*." (Dkt. No. 56-17.) After updating exposure categories from the interim report of the Goodyear plant, the authors confirmed "that workers in this plant have an increased risk of bladder cancer." (*Id.* at 3.) In the new study, 962 out of the 1,749 total Goodyear workers were classified as "definitely exposed." Of the 962 workers, 11 cases of bladder cancer were observed compared to an expected 1.88 cases, leading to an SIR value of 5.84. (*Id.* at 4.) Breaking down the 962 workers further and focusing on workers employed at Goodyear for fewer than five years, one reported case of bladder cancer compared to 0.8 epidemiologically expected cases yielded an SIR value of 1.25. (*Id.*) The authors had a separate categorization that, for 634 workers who had seen at least 20 years passed since their first employment at Goodyear, 8 cases of bladder cancer compared to an epidemiologically expected 0.89 yielded an SIR value of 9.02. (*Id.*) This 2010 study was updated in 2012 to reflect two changes: IARC's classification of OT as carcinogenic to humans and reports of 19 additional bladder cancer cases among the cohort of Goodyear workers studied. (Dkt. No. 56-18.) In the updated study, the jobs at the Goodyear plant were placed on a 10-point scale that reflected the severity of exposure to OT. For the 1970s, the three jobs that James held—lift truck operator, lab technician, and production operator—were assigned exposure values of 3, 5, and 10, respectively, with 10 being the highest possible score. (*Id.* at 11.) In a new classification that the authors would use for future updates of the Goodyear cohort, workers classified as "Definitely exposed moderate/high and regularly (DER)"—such as James—had a median of only 0.92 years in the highest category of exposure and yet had a strong correlation to overall exposure. (*Id.* at 13.) In

a 2013 update, the authors concluded that "[b]ladder cancer incidence remains elevated in this cohort and significantly associated with estimated cumulative exposure. Results are consistent with earlier findings in this and other cohorts. Despite other concurrent chemical exposures, we consider [OT] *most likely responsible for the bladder cancer incidence elevation and recommend a re-examination of occupational exposure limits*." (Dkt. No. 56-19 at 2 (emphasis added).) Among other information, the data in the 2013 update show a revised SIR value of 1.98 for definitely exposed workers employed for fewer than five years; and a revised SIR value of 3.974 workers who saw over 30 years passed since their first exposure. (*Id.* at 6.) The National Toxicology Program ("NTP") published a 13th Report on Carcinogens in 2014 that contained the following paragraph:

> Overall, there is credible evidence of an association between increased urinary-bladder cancer risk and exposure to *o*-toluidine based on consistent findings across studies, the relationship of cancer risk to exposure level and duration, and large magnitudes of effect across studies. An increased risk of urinary-bladder cancer (incidence or mortality) was observed in all four studies with adequate latency (time since first exposure) that used statistical or other methods capable of detecting an association (Case and Pearson 1954, Sorahan 2008, Pira *et al.* 2010, Carreón *et al.* 2014). Two studies did not find an excess of urinary-bladder cancer among *o*-toluidine–exposed workers; however, the statistical power to detect an effect was very low in the U.S. dye-workers study (Ott and Langner 1983), and misclassification of exposure was a serious concern for both the U.S. dye-workers cohort study and the Canadian case-control study (Richardson *et al.* 2007).

(Dkt. No. 58-12 at 3.) Other organizations have weighed in as well. For example, the American Conference of Governmental Industrial Hygienists issued a bulletin in 2001 that cited numerous studies concerning the damage that OT can cause to DNA as a prelude to cancer growth:

> Using a modified *E. coli* DNA repair test (pol A), 0.5 µg/ml o-toluidlne HCI was mutagenic in the absence of a metabolic activation system. In several Rec assays using *Bacillus subtillis* or *E. coli* strains, negative and positive results were reported. Studies in several yeast strains for mitotic recombinations, aneuploidy, and DNA repair showed positive results in most tests. The unscheduled DNA synthesis of HeLa cells and primary hepatocytes from rats and hamsters were increased by o-toluidine. In another report, the UDS in rat hepatocytes was not changed by treatment with o-toluidine. No increase in single-strand breaks could be observed in the DNA of V79 Chinese hamster lung fibroblasts incubated with 10 mmol

> o-toluidine for 2 hours in the presence of Aroclor-induced rat liver microsomes. An intraperitoneal injection of 100 mg/kg body weight produced single-strand DNA breaks in liver and kidney cells of male mice. Oral intubation of 200 mg/kg body weight inhibited testicular DNA synthesis in male mice, and intraperitoneal injection of lethal doses inhibited the renal DNA synthesis in suckling mice.

(Dkt. No. 58-14 at 3.)

E. This Case

Plaintiffs commenced this case by filing their complaint on July 14, 2017. (Dkt. No. 1.) In the complaint, plaintiffs cite concerns that DuPont and others had about OT and bladder cancer going back to the 1920s. After reciting James's work history, plaintiffs proceed to set forth three claims against defendants. In the first claim, plaintiffs accuse defendants of negligence "in researching, testing, manufacturing, marketing, supplying, promoting, packaging, labeling, selling, distributing, and in conducting their product stewardship of ortho-toluidine." (*Id.* at 18–19.) According to plaintiffs, defendants "failed to adequately and timely warn of the serious health hazards, including bladder cancer, associated with occupational exposure to ortho-toluidine." (*Id.* at 19.) In the second claim, plaintiffs accuse defendants of strict products liability because they were "engaged in the business of manufacturing and/or distributing ortho-toluidine which, without substantial change in its condition after it was sold, was the producing cause of the health problems of the Plaintiff and his aforementioned damages" even when OT was used "in a manner that was normally intended and reasonably anticipated by the Defendants." (*Id.* at 20–21.) In the third claim, Deborah seeks compensation for loss of consortium due to James's bladder cancer. Plaintiffs also seek punitive damages.

F. Plaintiffs' Expert Opinions

To support their claims, plaintiffs retained Dr. Oliver as a medical expert to address specific causation. Dr. Oliver prepared a report on June 2, 2018. (Dkt. No. 56-8.) Dr. Oliver reviewed the

literature cited above as well as James's medical records and other materials in the case. Dr. Oliver is a practicing physician who is board-certified in occupational and internal medicine. (*Id.* at 4.) Dr. Oliver has conducted research on pulmonary toxins and has conducted epidemiologic studies of working populations. (*Id.* at 5.) Dr. Oliver has worked with labor unions to identify potential health hazards and has testified before Congress and OSHA about those hazards. (*Id.*) Dr. Oliver also has an unusual credential compared to other experts whom plaintiffs could have consulted. In March 1979, Dr. Oliver directly observed Department 245 at the Goodyear plant when she conducted a walk-through inspection to evaluate reports of chest pain by workers in that department. (*Id.* at 3.)

As part of her medical opinion in this case, Dr. Oliver used differential etiology to rule out other factors known to be related to bladder cancer. Cigarette smoke is recognized as a risk factor for bladder cancer, and Dr. Oliver confirmed that James never has smoked. Benign prostatic hypertrophy (BPH) is associated with bladder cancer, but Dr. Oliver confirmed that James has no such symptoms. Obesity has been linked to an increased risk for bladder cancer; Dr. Oliver confirmed that James has been medically overweight but not obese. (*Id.* at 14.) Infection with polyoma virus has been linked to increased risk for bladder cancer. Dr. Oliver noted that James was not diagnosed with an infection of the virus and that a possible viral presence was not discussed until atypical cells already started appearing in cystoscopy results in 2012. (*Id.* at 15.) Dr. Oliver next reviewed James's likely exposure to other chemicals at Goodyear and to diesel fumes over the course of his trucking career. Dr. Oliver reviewed studies indicating that other chemicals had inconclusive associations with bladder cancer while OT "appears to be a potent human bladder carcinogen." (*Id.* at 16 (citation omitted).) Dr. Oliver reviewed studies of diesel fume exposure and concluded that, for James's level of outdoor exposure, any association with bladder cancer was inconclusive. (*Id.* at 17.) Finally, Dr. Oliver concluded that James had no family history, and no

personal history of chronic bladder infections or analgesic abuse, that would create other potential risk factors for bladder cancer. (*Id.* at 25.) The factors that Dr. Oliver considered and ruled out correspond to the external risk factors for bladder cancer considered in medical treatises on urology. "The primary culprits [of bladder cancer] are the aromatic amines that bind to DNA." (Dkt. No. 70-4 at 4.) Other external risk factors are heredity, smoking, nutritional factors, artificial sweeteners, analgesic abuse, and inflammation or infection. (*Id.*)

In the final part of her report, Dr. Oliver reviewed the literature concerning OT as a human bladder carcinogen. Dr. Oliver noted classifications by IARC and the NTP that OT was a known human carcinogen "based on sufficient evidence of carcinogenicity from studies showing that it causes urinary bladder cancer in humans." (*Id.* at 18 (citation omitted).) Dr. Oliver reviewed the original Goodyear study and the subsequent updates that the Court cited above. Dr. Oliver also reviewed animal studies in the literature and studies of a cohort of rubber chemical workers in the United Kingdom. (*Id.* at 19.) Dr. Oliver cross-referenced the literature with James's exposure to OT, most of which occurred at the highest levels designated in the literature and nearly all of which occurred with minimal respiratory and other protection. (*Id.* at 21.) Dr. Oliver further cited EPA guidelines suggesting that linear extrapolation of known data would be appropriate for mutagenic chemicals like OT. (*Id.* at 22.) Dr. Oliver ultimately concluded that no risk-free exposure to OT exists and that OT was a substantial contributing factor to James's bladder cancer. (*Id.*) Dr. Oliver's report includes 54 citations to various studies and governmental reports that have built up the literature over the years concerning OT and bladder cancer. (*Id.* at 29–31.)

Plaintiffs also retained Dr. Ronald Melnick as an expert to address general causation. Dr. Melnick is a PhD toxicologist who spent nearly 30 years as a senior toxicologist, project officer, and Director of Special Programs in the NTP. In his expert report (Dkt. No. 56-24), Dr. Melnick

explained that "[t]he most obvious reason for why animal models are used to evaluate human cancer risk is that it is unethical to test for adverse health effects such as cancer in humans through controlled intentional exposures." (*Id.* at 8.) Dr. Melnick explained that public health agencies rely on animal cancer data because

> all known human carcinogens that have been studied adequately in experimental animals produce positive carcinogenic results. Hence, even in the absence of adequate human data, public health agencies (IARC, 2006; NTP, 2014b; US EPA, 2005) have classified agents as possibly/probably or likely to be carcinogenic to humans or reasonably anticipated to be a human carcinogen if there is sufficient evidence in animals demonstrating either a) increased incidence of malignant or malignant and benign tumors combined in two or more species or at multiple sites, or b) increased incidence in two or more independent studies in one species, or c) increased incidence in a single study in one species if malignant tumors occur to an unusual degree in incidence, site, type, or age of onset. Several agents that were considered to be possible human carcinogens based on animal data were later confirmed as human carcinogens when reliable epidemiology data (usually occupational exposures) became available, e.g., 1,3-butadiene, cadmium, diethylstilbestrol, ethylene oxide, formaldehyde, vinyl chloride (Huff, 1993). *o*-Toluidine can be added to this list of chemicals that were found to be carcinogenic in experimental animals and later confirmed as a human carcinogen in subsequent epidemiological studies. Mechanistic data also contributes to evaluations of human cancer risk (IARC, 2006; NTP, 2014b; US EPA, 2005). Mechanistic data "help in assessing the relevance and importance of findings of cancer in animals and in humans" (IARC, 2006).

(*Id.* at 10–11.) Dr. Melnick then reviewed the literature addressing significant increases in tumor occurrences in rats after exposures to OT. (*Id.* at 15–16.) Next, Dr. Melnick surveyed the literature regarding the metabolism of toxins in general and aromatic amines in particular. According to the literature, aromatic amines break down when ingested and form metabolites that are known to be DNA-reactive; *i.e.*, these metabolites are known to have the ability to bind to DNA and to disrupt or to alter cellular function and reproduction. (*Id.* at 20–22.) These metabolites can be detected in the bloodstream when they react with hemoglobin to form compounds called hemoglobin adducts. Animal studies have shown that rats given a single dose of OT had detectable levels of metabolites in their urine and within six hours. (*Id.* at 24.) Workers at the Goodyear plant who were exposed to

OT had hemoglobin adducts of OT in their blood at levels significantly higher than workers who were not exposed to OT. (*Id.* at 25.) Studies have shown that humans developed hemoglobin adducts of OT when they received subcutaneous injections of an anesthetic called prilocaine that was metabolized to OT. (*Id.* at 25, 30.) Citing studies of the association between DNA damage and cancer, Dr. Melnick explained that DNA-reactive metabolites are significant because alterations in cellular DNA can cause cells to acquire "cancer capabilities" such as limitless replication, tissue invasion, and metastasis. (*Id.* at 28.) Animal studies have shown how metabolites of OT can bind to DNA at specific positions. (*Id.* at 30.) Other studies of animal exposure and bacterial metabolism have shown how metabolites of OT can create a genomic instability through events like DNA strand breaks that are never properly repaired. (*Id.* at 32; *see also* Dkt. No. 58-9 at 2.)

Defendants have attacked the opinions of plaintiffs' experts. Defendants note that animal studies often involve routes of exposure that do not reflect how humans would be exposed. (Dkt. No. 56-12 at 3.) Whether substances found to produce cancer in animals always produce cancer in humans is "a very difficult question to answer because in the cases where they apparently don't, it's usually because we're not completely sure that they don't in people. So the answer is there are many, many cases where they do. There are very few cases where we are fairly confident that they don't, and there are many cases where they appear not to, and we're still not sure." (*Id.* at 4.) Defense expert Dr. John O'Donoghue emphasized the limitations of animal studies when he noted in his report that "the possibility that a chemical that causes cancer in experimental animals may do so by a mechanism that does not occur in humans needs to be taken into account." (Dkt. No. 56-20 at 3.) Dr. O'Donoghue criticized animal studies of OT prior to 1978 because they did not always find bladder tumors in rats and mice, even when other tumors developed. (*Id.* at 5.) Dr. O'Donoghue asserted that there are "no reliable animal cancer bioassays conducted by applying either o-toluidine

or o-toluidine hydrochloride to the skin or by inhalation of their aerosols, vapors, or dusts." (*Id.* at 6.) Dr. O'Donoghue emphasized that the studies of the Goodyear plant limited the lowest category of exposure to less than five years of employment at the plant; this means, in his view, that the most extensive available study of OT at the Goodyear plant can offer no conclusive information about James's total of 996 unit-days of exposure. (*Id.* at 9.)

Dr. Oliver submitted a rebuttal report on October 5, 2018. (Dkt. No. 56-22.) To the extent that Dr. O'Donoghue also considered diesel fumes as a possible cause of James's bladder cancer, Dr. Oliver noted that studies of diesel fumes found associations with bladder cancer only for 10 years or more of exposure at high concentrations. (Dkt. No. 56-22 at 4.) Those same studies would have placed James at only moderate exposure. To the extent that Dr. O'Donoghue raised questions about polyoma virus and bladder cancer, Dr. Oliver responded that James had nothing in his medical history suggesting any chronic infection with polyoma virus. (*Id.* at 7.) Finally, Dr. Oliver responded to contentions from defendants that James's bladder cancer might have been caused by exposure to ionizing radiation from a time that he spent working for Union Carbide in Niagara Falls, New York. Dr. Oliver confirmed that James never worked in the areas of the facility that would have generated the most radiation exposure and that any exposure would have been minimal. (*Id.* at 9.)

Under questioning at her deposition, Dr. Oliver acknowledged that no one at Goodyear in 1974 took daily air, blood, or urine samples that would have given an exact, quantitative measurement of the extent to which James was exposed to OT. (Dkt. No. 56-23 at 21, 30.) Dr. Oliver did note that in 1990, Goodyear measured OT exposure at the sparkler filters and found a range between 1,500 and 3,600 ppm. (*Id.* at 24.) Dr. Oliver also asserted "that exposures that were measured in 1990 were in all likelihood lower than exposures that Mr. Sarkees experienced, because he experienced his exposures in 1974, and these exposures were measured in 1990. And between

1974 and 1990 there were engineering changes that were made at Goodyear which reduced the levels of exposure." (*Id.* at 27–28.) Dr. Oliver was asked about the data in the 2013 Goodyear update showing a revised SIR value of 1.98 for definitely exposed workers employed for fewer than five years. Because the confidence interval for that statistic included a value of 1.0, Dr. Oliver acknowledged that "strictly speaking, it is not statistically significant." (*Id.* at 68.) Dr. Oliver conceded that she had no specific information for other individuals in the Goodyear cohort, including what other exposures they may have had in their lives. (*Id.* at 71.) With respect to James's qualitative experiences with OT fumes, Dr. Oliver agreed that "you cannot quantify an exposure to a chemical merely by smelling it." (*Id.* at 84.)

G. Pending Motions

On July 26, 2019, defendants filed their motion for summary judgment and (alternatively) for preclusion of Dr. Oliver's testimony from trial. With respect to summary judgment or preclusion as against Dr. Oliver, defendants argue that "plaintiffs cannot prove that Mr. Sarkees had sufficient exposure to OT to cause his bladder cancer" and that "there is simply too great an analytical gap between the studies Dr. Oliver relies on and her conclusions." (Dkt. No. 56-1 at 25.) Defendants assert that "[t]here is no OT monitoring data for Mr. Sarkees' person or work areas while he was employed at Goodyear in 1974. Thus, no one knows Mr. Sarkees' actual level of exposure to OT." (*Id.* at 25–26; *see also* Dkt. No. 75 at 12, 16.) Defendant's fault plaintiffs and Dr. Oliver for ignoring that NIOSH "could not exclude aniline as a potential carcinogen" and that NIOSH hesitated to apply cancer risks for groups of Goodyear workers to individual workers. (Dkt. No. 56-1 at 26–27.) Based on exposure rankings for James's jobs and their calculation of his unit-days of exposure, defendants argue that "there is no statistically significant association between Mr. Sarkees' level of exposure to OT and bladder cancer, and thus there is no basis for conducting further causation

analysis, let alone finding that Mr. Sarkees' level of exposure to OT causes bladder cancer." (*Id.* at 29–30.) Defendants point out that one of the SIR values in one of the Goodyear studies has a confidence interval that reaches into the range of statistical insignificance; and that James was one year older at diagnosis than the cutoff age of 60 for another statistic from the studies about Goodyear workers. (*Id.* at 30–31; *see also* Dkt. No. 75 at 12.) Defendants assert that studies of short exposures to aromatic amines other than OT, or to other chemicals, are inapplicable and do not establish that short exposures can increase cancer risk for OT. (Dkt. No. 56-1 at 35–36.) Defendants emphasize that much of James's exposure was to Nailax—which contains chemicals besides OT—and not pure OT, further complicating efforts to quantify his actual exposure. (*Id.* at 40–41.) Defendants conclude their criticism of Dr. Oliver by arguing that she did not reliably rule out exposure to OT at Dunlop Tire. (*Id.* at 44–45.)

Defendants also argue several other points in favor of summary judgment. Defendants criticize plaintiffs' claim that they failed to exercise proper "product stewardship." Defendants argue that New York and Second Circuit law do not recognize a duty of product stewardship. "Assuming plaintiffs take the same position as in [another case by plaintiffs' counsel], plaintiffs' product stewardship claim boils down to a traditional failure to warn Goodyear of OT's hazards." (*Id.* at 46–47.) Defendants assert that plaintiffs' claim for punitive damages would fail if the claims for negligence and strict products liability also fail; alternatively, the claim for punitive damages would fail because the "lack of cancer warnings for OT in 1974 was consistent with the state of scientific knowledge at the time, during which OT's potential carcinogenicity was, at best, uncertain." (*Id.* at 51.) In defendants' view, a classification of OT as a carcinogen that came years after James's employment at Goodyear could not support the level of willful or wanton conduct needed to establish punitive damages. Finally, defendants argue against Deborah's claim for loss of

consortium because it is derivative of other claims that, in their view, necessitate summary judgment. Defendants argue further that Deborah cannot claim loss of consortium for an alleged toxic exposure that occurred 12 years before she married James.

Plaintiffs oppose the pending motions in all respects. Plaintiffs first defend Dr. Oliver's differential etiology. According to plaintiffs, Dr. Oliver properly excluded age and latency by citing studies indicating that most bladder cancer patients are 65 years of age or older and that bladder cancer risk is highest over 40 years after exposure. (Dkt. No. 70 at 13.) The parties do not dispute that tobacco usage is a risk factor and that James never used tobacco. (*Id.* at 14.) Plaintiffs note that some organizations set a "permissible" exposure limit for OT prior to its classification as a carcinogen; nonetheless, no organization ever has established a "safe" exposure limit, and NIOSH policy is that carcinogens do not have safe levels of exposure. (*Id.* at 15; *see also* Dkt. No. 70-6 at 21 ("Underlying this policy [of classification] is the recognition that there is no known safe level of exposure to a carcinogen, and therefore that reduction of worker exposure to chemical carcinogens as much as possible through elimination or substitution and engineering controls is the primary way to prevent occupational cancer. Accordingly, this policy no longer uses the term *recommended exposure limit (REL)* for chemical carcinogens; rather NIOSH will only recommend an initial starting point for control, called the Risk Management Limit for Carcinogens (RML-CA).").) Plaintiffs indicate that studies of the Goodyear plant probably underestimated the extent of James's exposure; air exposure was not measured in 1974 but only years later, and James was a 19-year-old temporary worker whose duties and safeguards did not follow the collective bargaining agreement with the local union. (Dkt. No. 70 at 18; *see also id.* at 23 ("It should be noted that ortho-toluidine exposure at Goodyear was significantly higher in 1974 than when studied by NIOSH in 1990. Between 1975 and 1987, six 'changes or procedures' were implemented which would have lowered exposure to

ortho-toluidine.").) Plaintiffs argue that the "NIOSH unit-day calculations are not sharp demarcations between 'safe' and injurious exposure" and that "even at the lowest exposure groupings in the Carreón study, there was a finding of an excess risk of bladder cancer." (*Id.* at 26, 27.) Overall, plaintiffs summarize 16 potential factors that Dr. Oliver reviewed and excluded before reaching her conclusions. (*Id.* at 40–41.)

Plaintiff next defend Dr. Melnick's opinions and discussion of cancer mechanisms. Plaintiffs emphasize the classification of OT as a carcinogen by multiple agencies, based partly on working group meetings in which Dr. Melnick participated. (*Id.* at 46.) Plaintiffs defend Dr. Melnick's assessment of animal studies because the toxicological findings from them can provide biological plausibility. (*Id.* at 47–50.)

With respect to the claims in the complaint, plaintiffs defend their view of product stewardship as part of an overall picture of negligence; Goodyear, in their view, failed to conduct sufficient monitoring at a time when, even with more definitive studies years away, suspicions about OT's carcinogenicity already existed. (*Id.* at 51–55.) In fact, plaintiffs assert that by 1974, defendants would have been aware of animal studies from the 1950s implicating OT as a bladder carcinogen. (*Id.* at 58–59.) Plaintiffs assert further that defendants always knew that any attempts at defining permissible exposure limits concerned only acute toxicity and "had nothing to do with protecting against cancer." (*Id.* at 61.) With respect to Deborah's claim for consortium, plaintiffs assert that current state law sets the accrual date of a toxic-exposure claim as the date of discovery, meaning that James discovered his bladder cancer well into his marriage with Deborah. (*Id.* at 67.)

III. DISCUSSION

The Court will begin its analysis by addressing the reliability of the opinions that Dr. Oliver and Dr. Melnick have expressed. The Court takes this approach for two reasons. Defendants' two motions include explicit requests to preclude plaintiffs' experts from testifying at trial. Also, the assessment of expert reliability will have a secondary effect on defendants' motion for summary judgment. As the Court explained previously to the parties (Dkt. No. 65), Rule 56(c)(4) requires both sides to "set out facts that would be admissible in evidence." Through the extensive exhibits attached to the motion papers, the parties have invoked their right to "object that the material cited to support or dispute a fact cannot be presented in a form that would be admissible in evidence." Fed. R. Civ. P. 56(c)(2).

A. Expert Reliability Generally

> *If it disagrees with experiment, it's wrong. In that simple statement is the key to science. It doesn't make a difference how beautiful your guess is, it doesn't make a difference how smart you are, who made the guess, or what his name is—if it disagrees with experiment, it's wrong. That's all there is to it.*[3]

Questions about expert admissibility begin with Rule of Evidence 702, which permits expert testimony if "(a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case." Fed. R. Evid. 702. "Of course, it would be unreasonable to conclude that the subject of scientific testimony must be 'known' to a certainty; arguably, there are no certainties in science. But, in order to qualify as

[3] Richard Feynman, Cornell University lecture (1964), *in* Robert Krulwich, *The Essence of Science Explained in 63 Seconds* (NPR May 17, 2012), https://www.npr.org/sections/krulwich/2012/05/17/152913171/the-essence-of-science-explained-in-63-seconds (last visited Feb. 25, 2020).

'scientific knowledge,' an inference or assertion must be derived by the scientific method. Proposed testimony must be supported by appropriate validation—*i.e.*, 'good grounds,' based on what is known. In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 590 (1993) (citations omitted). Many factors can affect scientific reliability, and no exhaustive list exists, but pertinent considerations can include the ability to test a theory or technique; the ability to subject a theory or technique to peer review and publication; the ability to determine the potential rate of error for a particular scientific technique; the existence and maintenance of standards controlling the technique's operation; and general acceptance of a particular theory or technique. *Id.* at 593–94 (citations omitted). In reviewing any pertinent considerations, courts have to make a distinction between the reasonableness of a theory or technique in general and the "particular method of analyzing the data thereby obtained, to draw a conclusion regarding *the particular matter to which the expert testimony was directly relevant*." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 154 (1999). Judges also have to watch for novelty driven by litigation; courts do not recognize any theory of chronic remunerative toxicity. Specifically, courts have to watch to see "whether the expert is proposing to testify about matters growing naturally and directly out of research they have conducted independent of the litigation, or whether they have developed their opinions expressly for the purposes of testifying." *Krause v. CSX Transp.*, 984 F. Supp. 2d 62, 75 (N.D.N.Y. 2013) (internal quotation marks and citations omitted); c*f. Lust By & Through Lust v. Merrell Dow Pharm., Inc.*, 89 F.3d 594, 597 (9th Cir. 1996) (affirming exclusion of "a professional plaintiff's witness" whose "chief premise was that if there is evidence of a positive association between an agent and a wide variety of birth defects in human epidemiological and animal studies, then the agent substantially increases the probability of all types of birth defects"); *Wills v. Amerada Hess Corp.*, No. 98 CIV. 7126 (RPP), 2002 WL 140542, at

*10 (S.D.N.Y. Jan. 31, 2002) ("The paucity of support for his opinion in his First Report demonstrates that Dr. Bidanset was ready to form a conclusion first, without any basis, and then try to justify it. He appears to be claiming that he is using the dose-response theory to conclude Decedent was 'intensely exposed to these carcinogenic hydrocarbons' and yet he makes no attempt to quantify Decedent's level of exposure, the essential step in using the dose-response theory."). After all, "the courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it." *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996); *see also Colon v. Abbott Labs.*, 397 F. Supp. 2d 405, 415 (E.D.N.Y. 2005) (rejecting an expert opinion in part because it was not tested and was "set forth for the first time in his April 2005 affidavit").

A reliability hearing always is an option but is unnecessary where, as here, the parties already have developed a full evidentiary record regarding the proposed expert opinions. "In this case, the parties have extensively briefed the issues pertinent to each expert's testimony. Each of the challenged experts has been subject to a lengthy deposition. Each of the expert's reports (as well as each of the reports of experts challenging the reliability of some of those reports) has been submitted to the court. It is difficult to think of anything missing from the presentation by the parties that could be pertinent to these in limine motions." *Malletier v. Dooney & Bourke, Inc.*, 525 F. Supp. 2d 558, 582 (S.D.N.Y. 2007).

As important as the gatekeeping function has become since *Daubert*, courts have to maintain the critical distinction between the threshold determination of reliability and the jury determination of weight. The threshold determination of reliability "does not mean that plaintiffs have to prove their case twice—they do not have to demonstrate to the judge by a preponderance of the evidence that the assessments of their experts are *correct*, they only have to demonstrate by a preponderance of evidence that their opinions are reliable . . . . The grounds for the expert's opinion merely have to be

good, they do not have to be perfect. The judge might think that there are good grounds for an expert's conclusion even if the judge thinks that there are better grounds for some alternative conclusion, and even if the judge thinks that a scientist's methodology has some flaws such that if they had been corrected, the scientist would have reached a different result." *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 744 (3d Cir. 1994) (citations omitted); *accord Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 267 (2d Cir. 2002) ("This limitation on when evidence should be excluded accords with the liberal admissibility standards of the federal rules and recognizes that our adversary system provides the necessary tools for challenging reliable, albeit debatable, expert testimony.") "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence. Additionally, in the event the trial court concludes that the scintilla of evidence presented supporting a position is insufficient to allow a reasonable juror to conclude that the position more likely than not is true, the court remains free to direct a judgment, Fed. Rule Civ. Proc. 50(a), and likewise to grant summary judgment." *Daubert*, 509 U.S. at 596 (citations omitted). "In short, *Daubert* neither requires nor empowers trial courts to determine which of several competing scientific theories has the best provenance. It demands only that the proponent of the evidence show that the expert's conclusion has been arrived at in a scientifically sound and methodologically reliable fashion." *Ruiz-Troche v. Pepsi Cola of Puerto Rico Bottling Co.*, 161 F.3d 77, 85 (1st Cir. 1998) (citations omitted).

Experts who seek to testify at trial also must establish their qualifications. "The initial question of whether a witness is qualified to be an 'expert' is important, among other reasons, because an 'expert' witness is permitted substantially more leeway than 'lay' witnesses in testifying as to opinions that are not rationally based on his or her perception." *Nimely v. City of New York*, 414

F.3d 381, 396 n.11 (2d Cir. 2005) (internal quotation and editorial marks and citations omitted). "Courts in this district often describe the analysis of an expert's qualifications as comprising two subparts. First, the court must examine the totality of the witness's background to determine whether he or she exhibits any one or more of the qualifications listed in Rule 702—knowledge, skill, experience, training, or education—with respect to a relevant field. Second, the court compares the area in which the witness has superior knowledge, education, experience, or skill with the subject matter of the proffered testimony." *Washington v. Kellwood Co.*, 105 F. Supp. 3d 293, 304 (S.D.N.Y. 2015) (internal quotation and editorial marks and citations omitted). Courts should not, however, exclude expert testimony simply because they do not "deem the proposed expert to be the best qualified or because the proposed expert does not have the specialization that the court[s] consider[] most appropriate." *Pineda v. Ford Motor Co.*, 520 F.3d 237, 244 (3d Cir. 2008) (internal quotation marks and citation omitted).

Ultimately, "[t]he party proffering the expert has the burden to demonstrate by a preponderance of the evidence that its expert witness satisfies these criteria." *R.F.M.A.S., Inc. v. So*, 748 F. Supp. 2d 244, 253 (S.D.N.Y. 2010) (citations omitted).

B. Toxic Exposure and Medical Causation

In the field of toxic torts, once experts establish their qualifications, they have to submit a scientifically reliable explanation for both general and specific causation. *See Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114, 1120–21 (N.Y. 2006) ("It is well-established that an opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation).") (citations omitted). "General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether

a substance caused a particular individual's injury. Evidence concerning specific causation in toxic tort cases is admissible only as a follow-up to admissible general-causation evidence. Thus, there is a two-step process in examining the admissibility of causation evidence in toxic tort cases. First, the district court must determine whether there is general causation. Second, if it concludes that there is admissible general-causation evidence, the district court must determine whether there is admissible specific-causation evidence." *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351 (5th Cir. 2007) (citations omitted); *see also Amorgianos*, 303 F.3d at 268 (2d Cir. 2002) ("More specifically, to establish causation, [plaintiffs] must offer admissible expert testimony regarding both general causation, i.e., that xylene exposure can cause the type of ailments from which Amorgianos claims to suffer; and specific causation, i.e., that xylene exposure actually caused his alleged neurological problems."). "Sequence matters: a plaintiff must establish general causation before moving to specific causation. Without the predicate proof of general causation, the tort claim fails." *In re Zoloft (Sertralinehydrochloride) Prod. Liab. Litig.*, 176 F. Supp. 3d 483, 491 (E.D. Pa. 2016) (internal quotation marks and citation omitted), *aff'd sub nom. In re Zoloft (Sertraline Hydrochloride) Prod. Liab. Litig.*, 858 F.3d 787 (3d Cir. 2017).

Ideally, general causation would be established by double-blind, randomized human clinical trials. *See In re Neurontin Mktg. & Sales Practices Litig.*, No. CIV.A. 04-CV10981PBS, 2010 WL 559108, at *1 (D. Mass. Feb. 12, 2010) ("DBRCTs [double-blind, randomized, controlled trials] are the 'gold standard' of scientific evidence. Experts must accord appropriate weights to different levels of evidence, i.e. a randomized, controlled trial, as the 'gold standard' of evidence, must be accorded greater weight than observational, non-controlled studies or case reports.") (citation omitted). As Dr. Melnick has pointed out in this case, however, there are obvious ethical reasons why double-blind, randomized human clinical trials rarely, if ever, will be available to establish

general causation for a potentially toxic substance. Experts alternatively will try to establish general causation by making reasonable inferences from other kinds of scientific information. *Compare St. Paul Fire & Marine Ins. Co. v. 111 Tenants Corp.*, 314 F. Supp. 2d 183, 186 (S.D.N.Y. 2003) (upholding an expert opinion reached "with reasonable scientific probability" and "supported by a rational process of elimination of alternative causes that is consistent with the findings of both experts") *with Riegel v. Medtronic, Inc.*, 451 F.3d 104, 127 (2d Cir. 2006) ("In this case, [the expert] essentially provided no explanation as to how he had reached his conclusion that the rupture must have been caused by a manufacturing defect, and himself seems to have backed away from this conclusion in his deposition. It was therefore appropriate for the district court to exclude his opinion."). Other kinds of scientific information can include microbial and animal clinical studies; case reports; and epidemiology studies. Among these non-clinical alternatives, "epidemiology is the best evidence of general causation in a toxic tort case. While the presence of epidemiology does not necessarily end the inquiry, where epidemiology is available, it cannot be ignored. As the best evidence of general causation, it must be addressed." *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) (citations omitted). All non-clinical alternatives, however, have to be assessed for their limitations, for any contradictory results, and for the inferences necessary to form an opinion about causation, because "[a] correlation does not equal causation." *Id.* at 885. "The specific way an expert conducts such an analysis must be reliable; all of the relevant evidence must be gathered, and the assessment or weighing of that evidence must not be arbitrary, but must itself be based on methods of science." *In re Zoloft (Sertraline Hydrochloride) Prod. Liab. Litig.*, 858 F.3d 787, 796 (3d Cir. 2017) (internal quotation marks and citation omitted). *Compare Pritchard v. Dow Agro Scis.*, 705 F. Supp. 2d 471, 488 (W.D. Pa. 2010) (precluding expert testimony where "there is no evidence of record explaining the method" used to link chlorpyrifos exposure and non-Hodgkin's lymphoma and where counsel

drafted the expert's attempt at explaining certain calculations) *and Wheat v. Sofamor, S.N.C.*, 46 F. Supp. 2d 1351, 1359 (N.D. Ga. 1999) ("[A]lthough the differential diagnosis method may be generally accepted and practiced, Plaintiffs failed to show that any other expert has reached the conclusion that the mere implantation of pedicle screws causes back injuries and pain. For these reasons, the Court finds [the expert's] testimony unreliable and inadmissible.") *with Lightfoot v. Georgia-Pac. Wood Prod., LLC*, No. 7:16-CV-244-FL, 2018 WL 4517616, at *21 (E.D.N.C. Sept. 20, 2018) ("Further, in addition to referencing directly studies cited in Dr. Aronson's report, Dr. Boles independently discusses studies supporting his general causation opinion, including Leivo (2016), Binazzi (2015,) and IARC (2012). (Boles Rep. 1-2). In sum, Dr. Boles's independent general causation opinion [about wood dust and sinonasal cancer] is both relevant and reliable."), *order amended on reconsideration*, No. 7:16-CV-244-FL, 2018 WL 6729636 (E.D.N.C. Dec. 21, 2018). A case involving exposure to ethylene oxide (ETO) and ethylene chlorohydrin (ECH) illustrated the kinds of details needed to raise an opinion about a "possible cause" of an injury to the level of scientific reliability:

> No published scientific literature reports mental retardation in any species at any dose level resulting from exposure to ETO or ECH. No expert designated to testify on plaintiffs' behalf in this action has submitted his or her conclusions for peer review that ETO and/or ECH can cause birth defects. There is no evidence of a statistically significant association between the ingestion of ETO-sterilized alfalfa tablets and mental retardation. No expert designated to testify on plaintiffs' behalf in this action can identify either the number of mutagenic agents or the amount of such agents to which plaintiffs' parents were exposed prior to plaintiffs' births. Each expert designated to testify on plaintiffs' behalf in this case has assumed that plaintiffs' parents were not exposed to any substances, other than ETO and/or ECH, which have the ability to produce either teratogenic or mutagenic effects. The theories of causation which will be advanced by the expert witnesses designated to testify on plaintiffs' behalf in this case cannot distinguish the effects of ETO or ECH from other mutagenic or teratogenic substances to which plaintiffs' parents may have been exposed. Cigarette smoke contains ETO, as well as numerous other alkylating agents.

Sorensen v. Shaklee Corp., No. CIV. 1-91-CV-70007, 1993 WL 735819, at *2 (S.D. Iowa Sept. 28, 1993).

If experts can establish general causation then they must proceed to establish specific causation:

> To establish specific causation, an expert must first complete the general causation analysis, and then must establish, at a minimum:
>
> • *Qualitative Toxicity.* The chemical in issue is known to be capable of producing the alleged effect in humans. In some instances, animal data or other information may be applied to the analysis and the biologic plausibility of such extrapolation must be evaluated in this assessment. [This is essentially the results of the general causation analysis.]
>
> • *Dose–Response.* The individual had contact with the chemical (exposure), and the amount of chemical absorbed into the body (dose) was of sufficient magnitude and duration to be capable of producing the alleged effect.
>
> • *Temporality.* The chemical exposure must be related in time to the onset of the individual's clinical condition, e.g., the effect did not precede the alleged exposure.
>
> • *Confounders.* All other significant causes (including exposure to other substances, lifestyle, workplace, and genetic factors) of the individual's clinical condition have been controlled for or ruled out. This is essentially the same procedure a physician performs when conducting a differential diagnosis.
>
> • *Coherence.* All of the evidence is consistent with the conclusion of causation.
>
> Failure to establish even one of these criteria for specific causation is usually fatal to the proposition that exposure to a specific chemical caused a specific medical condition, i.e., individual causation.

Downs v. Perstorp Components, Inc., 126 F. Supp. 2d 1090, 1095 (E.D. Tenn. 1999); *see also Zuchowicz v. United States*, 140 F.3d 381, 383 (2d Cir. 1998) ("There is, moreover, no older requirement in this area of law than the need to show such a link between the defendant's actions and the plaintiff's loss."); *Allen v. Pennsylvania Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) ("Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case.") (citation omitted). Some issues pertaining to the above factors are worth noting. Some injuries are "signature diseases": injuries unique to a single pathogen or chemical agent, such that the presence

of the injury is proof of the presence of the pathogen or agent. One well-known signature disease is mesothelioma, a cancer of the lungs reliably established in the literature as uniquely associated with exposure to asbestos. A "signature disease" theory requires ruling out other possible causes of an alleged injury. *Cf., e.g., Nat'l Bank of Commerce (of El Dorado, Ark.) v. Dow Chem. Co.*, 965 F. Supp. 1490, 1514–15 (E.D. Ark. 1996) (expert's "signature disease" theory precluded for failure to rule out other causes). With respect to measuring dose-response or amount of exposure, "exposure can be estimated through the use of mathematical modeling by taking a plaintiff's work history into account to estimate the exposure to a toxin. It is also possible that more qualitative means could be used to express a plaintiff's exposure. Comparison to the exposure levels of subjects of other studies could be helpful provided that the expert made a specific comparison sufficient to show how the plaintiff's exposure level related to those of the other subjects. These, along with others, could be potentially acceptable ways to demonstrate causation if they were found to be generally accepted as reliable in the scientific community." *Parker*, 857 N.E.2d at 1121. In contrast, a complete lack of quantitative information will not work. Experts will fail to establish specific causation where they rely entirely on qualitative descriptions such as "far more exposure than in the epidemiological studies" and "frequently exposed" to "excessive amounts," especially where epidemiological links between exposure and disease are weak or missing. *See id.* at 1122 ("Plaintiff's experts were unable to identify a single epidemiologic study finding an increased risk of AML as a result of exposure to gasoline. In addition, standards promulgated by regulatory agencies as protective measures are inadequate to demonstrate legal causation."); *see also Amorgianos*, 303 F.3d at 268 (expert properly excluded for failure to apply his own methodology reliably, where he identified numerous factors affecting exposure to xylene from paint but then calculated plaintiff's xylene exposure from only a few of those factors). Courts also have to make an important distinction between actual scientific

occurrence in the literature and theoretical plausibility. *See Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996) ("At a minimum, we think that there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered."); *accord Nat'l Bank of Commerce v. Associated Milk Producers, Inc.*, 22 F. Supp. 2d 942, 961 (E.D. Ark. 1998) ("The Court is of the opinion that the 'no-threshold' or 'one-shot' theory has respectable scientific support as a matter of scientific theory. But, ... it is not enough to show that a chemical agent 'sometimes causes the kind of harm complained of,' and here we know, that plaintiff has not even shown that AFM [aflatoxin M–1] 'sometimes causes' laryngeal cancer.").

C. General Causation and Dr. Melnick

The Court now will apply the above principles of scientific reliability to plaintiffs' experts. Plaintiffs retained Dr. Melnick to offer an opinion about general causation only. Dr. Melnick's qualifications are acceptable; he spent decades as a PhD toxicologist, and he "helped other study scientists design health effects studies and provided guidance on how to incorporate relevant mechanistic and pharmacokinetic studies into their overall research programs." (Dkt. No. 56-24 at 5.) As the Court noted above, Dr. Melnick reviewed the history of animal studies and how every currently known human carcinogen was first confirmed as an animal carcinogen through animal studies. That history of animal studies by itself would not offer any definitive proof about OT, but the list of human carcinogens successfully investigated with animal studies is long: 1,3-butadiene, cadmium, diethylstilbestrol, ethylene oxide, formaldehyde, and vinyl chloride. Decades of research went into the investigation of other known human carcinogens, and the body of research literature shows that animal studies at least can be informative absent human studies to the contrary. *Compare Metabolife Int'l, Inc. v. Wornick*, 264 F.3d 832, 842 (9th Cir. 2001) ("Notwithstanding the moral and

ethical problems often surrounding animal studies, in some circumstances they provide useful data about human health. The district court erred in rejecting the animal studies proffered by Metabolife merely because of the species gap.") *with Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1314 (11th Cir. 1999) ("Furthermore, Allison does not explain why the results of these animal studies should trump more than twenty controlled epidemiological studies of breast implants in humans which have found no valid increased risk of autoimmune disease.") (citations omitted). The animal studies involving OT should be placed in that context. NCI and other agencies have published numerous reports of animal studies over the years involving OT. Some studies have showed stronger results than others, but the overall body of literature for several decades has tended in the direction of showing a statistically significantly higher incidence of tumors, bladder cancer, or other cancers for animals exposed to OT. Significantly, the studies that Dr. Melnick discussed were not the type of case-control studies or meta-analyses—coupled with cohort studies lacking any documentation of exposure—that doomed the expert opinion in *Knight v. Kirby Inland Marine, Inc.*, 363 F. Supp. 2d 859, 865–66 (N.D. Miss. 2005); *see also Rider v. Sandoz Pharm. Corp.*, 295 F.3d 1194, 1199 (11th Cir. 2002) (excessive reliance on case reports that "report symptoms observed in a single patient in an uncontrolled context"); *Rizzo v. Applied Materials, Inc.*, No. 615CV557MADATB, 2017 WL 4005625, at *8 (N.D.N.Y. Sept. 11, 2017) (expert opinion precluded where it rested on no epidemiological studies and on animal testing with no conclusions regarding any specific disease). Dr. Melnick then reviewed the research literature to address the inferential steps by which researchers have proposed a likely mechanism for OT's mutagenic effects. The inferences proceed something like this. Animal studies have shown a higher incidence of cancer among animals exposed to OT. Animals exposed to OT are found to have hemoglobin adducts and other metabolites in their bodies that are suspected to be DNA-reactive. Since the basic structure and chemistry of DNA is the same in all

living organisms—differences in coding account for the rich diversity of life—researchers over the years have taken relatively simple and accessible bacterial DNA and exposed it to OT or OT metabolites. Researchers found that the bacterial DNA had strand breaks and other disruptions analogous to long-established literature about cancer genesis in humans:

> Although the mechanisms of carcinogenicity of o-toluidine are not completely understood, the available evidence suggests that they are complex and involve several key modes of action, including metabolic activation that results in binding of reactive metabolites to DNA and proteins, mutagenicity, oxidative DNA damage, chromosomal damage, and cytotoxicity (Skipper *et al.* 2010). The key metabolic activation steps and genotoxic effects occur in both experimental animals and humans.
>
> Metabolism of monocyclic aromatic amines, including *o*-toluidine, involves many competing activating and deactivating pathways, including N-acetylation, N-oxidation and N-hydroxylation, and ring oxidation. Cytochrome P450–mediated N-hydroxylation to Nhydroxy-*o*-toluidine, a carcinogenic metabolite, occurs in the liver. N-hydroxy-*o*-toluidine can be either metabolized to *o*-nitrosotoluene or conjugated with glucuronic acid or sulfate and transported to the urinary bladder via the circulation. Once in the urinary bladder, N-hydroxy-*o*-toluidine can be released from the conjugates in an acidic urine environment to either react directly with DNA or be bioactivated via sulfation or acetylation by cytosolic sulfotransferases or N-acetyltransferases (presumably NAT1). The postulated activated form (based on comparison with other aromatic amines), N-acetoxyo-toluidine, is a reactive ester that forms electrophilic arylnitrenium ions that can bind to DNA (Kadlubar and Badawi 1995, Riedel *et al.* 2006, English *et al.* 2012).

(Dkt. No. 58-12 at 4.) Studies of factory workers and of surgical patients receiving prilocaine showed that those workers and patients had the same chemicals in their blood or urine after exposure. With the research literature having taken this course so far, does that literature establish definitively that OT always and predictably will disrupt someone's DNA in a way that always will lead to cancer? Can the decades of research on OT and other mutagens function like the equivalent of an instant-replay camera that shows the exact moment when some molecules of OT entered James's body, broke his DNA strands, and created abnormal cells that led to cancer 42 years later? Of course not, and Dr. Melnick has not proposed anything nearly that aggressive. *Cf. Allen v.*

Pennsylvania Eng'g Corp., 102 F.3d 194, 198 (5th Cir. 1996) (affirming expert inadmissibility where, *inter alia*, "the cell biology data show only that EtO [ethylene oxide] has mutagenic and genotoxic capabilities in living organisms, not that it necessarily causes brain cancer in humans or in Allen's particular case"); *Bourne ex rel. Bourne v. E.I. Dupont de Nemours & Co.*, 189 F. Supp. 2d 482, 496 (S.D.W. Va. 2002) ("Courts considering the reliability of experts' extrapolation to human teratogenicity from *in vivo* and *in vitro* tests have recognized that such tests are generally considered to be suspect when relied upon for that purpose."). In fact, Dr. Melnick really has not proposed anything new at all. Based on decades of research involving OT, other mutagens, and likely mechanisms of DNA disruption, Dr. Melnick simply has tied together established inferences to propose that OT, once in a human body, likely has the capacity to alter DNA directly and through its metabolites. *Cf. Pub. Citizen Health Research Grp. v. Tyson*, 796 F.2d 1479, 1491 (D.C. Cir. 1986) (a "conclusion that mutagenic and cytogenic chemicals are carcinogenic" can support an inference of harmful health effects); *In re New York City Asbestos Litig.*, 39 N.Y.S.3d 629, 642 (N.Y. Sup. Ct. 2016) ("Unlike the benzine contained in gasoline at issue in *Parker*, plaintiffs observe that the connection between asbestos dust and mesothelioma is well known (providing the basis for general causation)."). Multiple governmental and industry agencies have agreed and have classified OT as a carcinogen. DuPont itself indirectly supported the portion of Dr. Melnick's opinion concerning binding of hemoglobin; in 1985, DuPont issued a warning that "[h]uman health effects of [OT] overexposure may initially include: reduction of the blood's oxygen-carrying capacity with cyanosis (bluish discoloration), weakness, or shortness of breath by formation of methemoglobin." (Dkt. No. 57-13 at 4.) Speculative techniques such as extrapolation are not needed here nearly as much as they were in other cases that led to expert preclusion. *Cf. In re Prempro Prod. Liab. Litig.*, 738 F. Supp. 2d 887, 894 (E.D. Ark. 2010) (precluding expert where "Dr. Aldaz has not explained how these

[animal] studies could be reliably extrapolated to predict the effects of Premarin in humans. And for the most part, he also did not elucidate the relationship between the estrogen metabolites in the studies and those substances actually found in Premarin."). To the extent that defendants object to the research literature, they "conflate the idea of *toxicity* with the idea of *causation*." *Todd v. Tempur-Sealy Int'l, Inc.*, No. 13-CV-04984-JST, 2016 WL 5462428, at *3 (N.D. Cal. Sept. 28, 2016) (finding that an expert opinion about carcinogens having no safe dose of exposure "is supported by a reputable treatise in the field of toxicology"); *but see Adams v. Cooper Indus., Inc.*, No. CIVA 03-476 JBC, 2007 WL 2219212, at *7 (E.D. Ky. July 30, 2007) (criticizing the "no-safe-dose" theory).

So long as Dr. Melnick avoids any commentary about specific causation and acknowledges the inferences needed in the current body of literature, his testimony about general causation is reliable and should be permitted at trial.

D. Specific Causation and Dr. Oliver

In a similar fashion, the Court next will apply the above principles of scientific reliability to Dr. Oliver's opinion about specific causation. The Court finds Dr. Oliver's credentials acceptable; she spent decades as a board-certified physician in occupational and environmental medicine, and she has the unique perspective of having inspected the Goodyear plant herself just a few years after James worked there, to help evaluate potential health hazards. Dr. Oliver candidly acknowledged two variables that create both positive and negative inferences for the amount of exposure that James received from OT. On the negative side, no one at Goodyear thought to measure air concentrations of OT in the areas where James worked in 1974, and no one thought to log instances of contaminated clothing and direct skin contact. The exact numerical amount of exposure that James received, therefore, will never be known. On the positive side, however, direct measurements began occurring by the late 1970s and the 1980s, and by that time, various safeguards pertaining to

protection and ventilation were in place that did not benefit James in 1974. Dr. Oliver thus inferred reasonably that the exposure levels measured in subsequent years are underestimates of the intensity of James's exposure. *Cf. Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 264 (4th Cir. 1999) ("Consequently, while precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff's exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial exposure and need not invariably provide the basis for an expert's opinion on causation."). The measurements from later years, coupled with the inference of underestimation, suffices to make the intensity of James's likely exposure much more specific than the descriptions in *Parker*, which contained no quantitative information at all. As for duration of exposure, Dr. Oliver reasonably focused on James's exposure to OT as opposed to other chemicals. Defendants have argued strenuously that Dr. Oliver has overestimated James's exposure to OT because his most extensive exposure was to Nailax, a blend of chemicals only one of which is OT. The Goodyear studies with their periodic updates, however, have been occurring for decades. Nowhere in the research literature or in Dr. Oliver's direct observations of the Goodyear plant is there any suggestion that the Nailax / Wing-Stay blend chemically altered OT in any way that would have altered the mutagenic properties that have been suggested throughout the literature. (*See, e.g.*, Dkt. No. 56-14 at 4.) Defendants' argument about Nailax versus OT would have had much more traction had Dr. Oliver relied on some simplistic calculation of her own that accounted only for total volume of Nailax. Dr. Oliver instead worked consistently with the Goodyear studies and worked with the assumption that OT retained its identity and properties in the Nailax blend and thus would have had its own partial pressure in the air and its own direct skin and clothing contact with James. Dr. Oliver's analysis does not require "too many extrapolations from dissimilar data, too many analytical

leaps and . . . a loose application of purportedly objective scientific causation standards." *Caraker v. Sandoz Pharm. Corp.*, 188 F. Supp. 2d 1026, 1031 (S.D. Ill. 2001).

The other aspect of duration of exposure that has drawn intense objection from defendants concerns the various classifications that evolved over the course of the Goodyear studies. Defendants make much of the lowest classification of duration that appears in the Goodyear studies: workers exposed to OT for under five years. In effect, defendants want to argue that James's position at the low end of that category—his maximum exposure was seven months—means that his place in the Goodyear studies and in other studies tails off into statistical ambiguity and insignificance. The Goodyear studies, however, are well-respected studies and reliably show indications of risk above any baseline population. Workers classified as "Definitely exposed moderate/high and regularly (DER)"—such as James—had a median of only 0.92 years in the highest category of exposure and yet had a strong correlation to overall exposure. The Goodyear study authors would have been aware of defendants' objection, yet concluded that "[b]ladder cancer incidence remains elevated in this cohort and significantly associated with estimated cumulative exposure. Results are consistent with earlier findings in this and other cohorts. Despite other concurrent chemical exposures, we consider [OT] *most likely responsible for the bladder cancer incidence elevation and recommend a re-examination of occupational exposure limits*." (Dkt. No. 56-19 at 2 (emphasis added).) Among other information, the data in the 2013 update show a revised SIR value of 1.98 for definitely exposed workers employed for fewer than five years; and a revised SIR value of 3.974 workers who saw over 30 years passed since their first exposure. (*Id.* at 6.) Defendants correctly point to one statistic whose confidence interval does dip into a range of statistical insignificance. Nonetheless,

> Under Rule 702, federal courts routinely permit witnesses with "technical or other specialized knowledge" to state opinions on matters where the data falls short

> of proving the witness's conclusion. For example, an art appraiser testifying about a painting's authenticity might state an opinion based in part on scientific analysis, but the ultimate conclusion would come from the witness's specialized knowledge, training and experience. Scientists, too, form professional opinions that are reasonably based on "good science" but where the data is insufficient for definitive scientific proof. To hold the opinions of scientists inadmissible unless backed by statistically significant results from tightly controlled (and very expensive) experiments would set a separate, higher standard for scientists than for other witnesses with specialized knowledge.

In re Ephedra Prod. Liab. Litig., 393 F. Supp. 2d 181, 188 (S.D.N.Y. 2005). The rest of the body of literature, additionally, shows reliable epidemiological suggestions that James received enough exposure to a likely mutagen—whose likely mechanism has no safe exposure limits—that the mutagen would have been capable of cellular changes that would manifest themselves as bladder cancer a generation later. James's situation thus differs from one case that defendants cited where "*not one* study has shown a statistically significant causal relationship between the vaccine and chronic joint pain." *Awad v. Merck & Co.*, 99 F. Supp. 2d 301, 307 (S.D.N.Y. 1999) (emphasis added). "One can never point to an individual person in a study and say that person's injury was definitely caused by the exposure." (Dkt. No. 56-1 at 36.) The Court agrees while also noting that *Daubert* and Rule 702 do not require such a high level of certainty. *Cf. Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1161–62 (E.D. Wash. 2009) ("All of the experts in this case agree benzene-induced diseases are dose dependent, and Gardner testified in his deposition that duration and frequency of exposure are important factors to consider. Yet, in his report, Gardner did not attempt to quantify dose or even estimate Henricksen's level of exposure to benzene. Implicitly, Gardner's opinion presumes that exposure to benzene in gasoline can cause AML [acute myelogenous leukemia] in any dose and that Henricksen's exposure was sufficient. Gardner's opinion is undermined by his failure to analyze or evaluate (his own or any other expert's) information pertaining to dose or the actual level of Henricksen's exposure. This renders his opinion on specific causation inherently unreliable.").

The final step in Dr. Oliver's analysis concerned differential etiology. As the Court noted above, Dr. Oliver identified numerous factors that can elevate risk of bladder cancer, including tobacco use, family history, obesity, polyoma virus infections, and other chemical exposures including diesel fumes. One by one, Dr. Oliver demonstrated that these other factors either were not present at all in James's life or would have had only a marginal impact on James's overall risk of developing bladder cancer. Dr. Oliver's efforts will suffice to establish differential etiology:

> A medical expert's opinion based upon differential diagnosis normally should not be excluded because the expert has failed to rule out every possible alternative cause of a plaintiff's illness. In such cases, the alternative causes suggested by a defendant normally affect the weight that the jury should give the expert's testimony and not the admissibility of that testimony. However, a differential diagnosis that fails to take serious account of other potential causes may be so lacking that it cannot provide a reliable basis for an opinion on causation. Thus, if an expert utterly fails to consider alternative causes or fails to offer an explanation for why the proffered alternative cause was not the sole cause, a district court is justified in excluding the expert's testimony.

Cooper v. Smith & Nephew, Inc., 259 F.3d 194, 202 (4th Cir. 2001) (internal quotation marks and citations omitted).

In raising questions about warnings and precautions that James might have ignored; the exact tasks that James might have performed in 1974; and other uncertainties about the measurement of workplace hazards at the Goodyear plant in the early 1970s, defendants have identified subjects that they can explore in their case in chief and on cross-examination at trial. The Court's review of plaintiffs' expert opinions is not intended to preclude any line of questioning at cross-examination. The Court's sole task here is to assess these threshold reliability of Dr. Oliver's work. Dr. Oliver has hewn close to the body of research literature concerning human exposure to OT and epidemiological assessment of subsequent cases of bladder cancer. Any inferences that Dr. Oliver has made were reasonable based on the literature. The Court consequently concludes that plaintiffs should be allowed to proceed with presenting Dr. Oliver's work to a jury.

E. Summary Judgment Generally

The Court's work reviewing plaintiffs' expert opinions addresses most of the arguments that defendants made for summary judgment. Assessment of the motion for summary judgment thus will be brief.

"The court shall grant summary judgment if the movant shows that there is no genuine dispute as to any material fact and the movant is entitled to judgment as a matter of law." Fed. R. Civ. P. 56(a). "As to materiality, the substantive law will identify which facts are material. Only disputes over facts that might affect the outcome of the suit under the governing law will properly preclude the entry of summary judgment . . . . More important for present purposes, summary judgment will not lie if the dispute about a material fact is 'genuine,' that is, if the evidence is such that a reasonable jury could return a verdict for the nonmoving party." *Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 248 (1986) (citation omitted). "The party seeking summary judgment has the burden to demonstrate that no genuine issue of material fact exists. In determining whether a genuine issue of material fact exists, a court must examine the evidence in the light most favorable to, and draw all inferences in favor of, the non-movant . . . . Summary judgment is improper if there is any evidence in the record that could reasonably support a jury's verdict for the non-moving party." *Marvel Characters, Inc. v. Simon*, 310 F.3d 280, 286 (2d Cir. 2002) (citations omitted). "Where, as here, the nonmovant would bear the burden of proof at trial, the movant may show prima facie entitlement to summary judgment by either (1) pointing to evidence that negates its opponent's claims or (2) identifying those portions of its opponent's evidence that demonstrate the absence of a genuine issue of material fact." *Barlow v. Male Geneva Police Officer who Arrested me on Jan. 2005*, 434 F. App'x 22, 25 (2d Cir. 2011) (summary order) (internal quotation and editorial marks and citation omitted).

F. Questions of Fact Requiring Trial

After reviewing the record, the Court notes that most of defendants' arguments for summary judgment derive from two sentences in their motion papers: "There is no OT monitoring data for Mr. Sarkees' person or work areas while he was employed at Goodyear in 1974. Thus, no one knows Mr. Sarkees' actual level of exposure to OT." (Dkt. No. 56-1 at 26.) These two sentences highlight the differences between determinations of scientific reliability and factual questions of negligence best left to a jury. Defendants are right that James's exact OT exposure in 1974 never will be reduced to a precise, accurate number. As discussed above, plaintiffs have presented a decades-old body of research literature to make the next best available argument: that OT likely is capable of producing bladder cancer in humans, based on inferences from reliable bacteriological, animal, and epidemiological studies; that factory workers with intense exposure to OT had some association with increased risk of bladder cancer over 20 years later, even when the duration of exposure was under five years; and that James likely encountered a threshold level of exposure based on qualitative assessments from 1974 and quantitative assessments a few years later. "By its nature, epidemiology is ill-suited to lead a factfinder toward definitive answers, dealing as it does in statistical probabilities and the continual possibility of confounding causal factors . . . . Applied to epidemiological studies, the question is not whether there is some dispute about the validity or force of a given study, but rather, whether it would be unreasonable for a rational jury to rely on that study to find causation by a preponderance of the evidence. In addition, multiple epidemiological studies cannot be evaluated in isolation from each other. Unlike admissibility assessments, which involve decisions about individual pieces of evidence, sufficiency assessments entail a review of the sum total of a plaintiff's evidence." *In re Joint E. & S. Dist. Asbestos Litig.*, 52 F.3d 1124, 1133 (2d Cir. 1995). Despite the scientific reliability of plaintiffs' inferences and

arguments, numerous questions of fact remain concerning James's day-to-day work assignments, safety training, and available safeguards. *Cf. Suter v. W.R. Grace & Co.-Conn.*, 599 N.Y.S.2d 260, 261 (N.Y. App. Div. 1993) ("Plaintiff's personal affidavit along with other evidence indicates that her late husband was likely exposed to asbestos-containing material which was manufactured by W.R. Grace. Accordingly, questions of fact are raised as to whether said exposure resulted in the death of plaintiff's husband allegedly caused by asbestos-related lung cancer."). Regardless of any studies or likelihoods or inferences that plaintiffs present, a jury has to resolve the ultimate question of whether OT more likely than not was a substantial contributing factor to James's bladder cancer. *See* N.Y. Pattern Jury Instr.—Civil 2:70 ("In most cases care should be taken to use the phrases 'a cause' and "a substantial factor" and to avoid the use of the phrases 'the cause' and 'that cause' in order to prevent the erroneous implication that there can be only one proximate cause.") (citations omitted). Regardless of any *Daubert* analysis, the jury remains free to weigh the credibility of plaintiffs and their experts. Defendants remain free to probe plaintiffs' expert opinions and James's subsequent work and social history, all through cross-examination. Resolving these numerous variables as a matter of law would be inappropriate.

Defendants' remaining arguments require only brief attention. Defendants argue that "there is no separate cause of action for 'product stewardship,' and that claim should be dismissed." (Dkt. No. 56-1 at 47.) The phrase "product stewardship" appears only twice in plaintiffs' complaint, in consecutive sentences: "Defendants failed to exercise reasonable care in researching, testing, manufacturing, marketing, supplying, promoting, packaging, labeling, selling, distributing, and in conducting their product stewardship of ortho-toluidine; [and] Defendants failed to exercise ordinary care and/or were reckless in conducting their product stewardship of ortho-toluidine and in researching, testing, manufacturing, marketing, supplying, promoting, packaging, labeling, selling,

and/or distributing ortho-toluidine into interstate commerce in that Defendants knew or should have known that occupational exposure to ortho-toluidine carried a risk of unreasonable and dangerous side effects, including bladder cancer." (Dkt. No. 1 at 18–19.) These two sentences, in turn, are surrounded in the complaint by other accusations about failure to warn and inadequate safety instructions, all as part of plaintiffs' first claim for common-law negligence. At most, then, "product stewardship" is one descriptive phrase among many that plaintiffs have advanced to explain their negligence claim. Plaintiffs have not advanced "product stewardship" as a novel legal theory of liability. *Cf. In re Methyl Tertiary Butyl Ether (MTBE) Prod. Liab. Litig.*, 643 F. Supp. 2d 482, 497 (S.D.N.Y. 2009) (precluding expert testimony about product stewardship because it involved "neither scientific technique nor technical expertise" but implicitly acknowledging it). Unless plaintiffs at trial insist on extensive testimony or some kind of jury charge specifically about product stewardship, defendants' request for summary judgment on any "claim" about product stewardship should be denied as moot. Defendants have requested summary judgment on plaintiffs' demand for punitive damages. The Court agrees with defendants generally that punitive damages require crossing a high threshold of reckless or wanton conduct. *See, e.g., Walker v. Sheldon*, 179 N.E.2d 497, 499 (N.Y. 1961) (citations omitted). Right now, though, the record contains questions of fact about James' day-to-day work environment at Goodyear and the extent to which workers were expected to deviate from any policies, training, warnings, or precautions that might have existed on paper. (*See, e.g.*, Dkt. No. 56-32 at 11–14; Dkt. No. 57-1 at 6 (1977 DuPont memo asserting that "we have seen no evidence that [OT] ever caused cancer in any of our employees").) *See Greenbaum v. Handelsbanken*, 67 F. Supp. 2d 228, 267 (S.D.N.Y. 1999) ("Under New York law, whether to award punitive damages and how much to award are primarily questions which reside in the sound discretion of the original trier of the facts.") (internal quotation marks and citation omitted); *O'Neill*

v. Yield House Inc., 892 F. Supp. 76, 78–79 (S.D.N.Y. 1995) (jury did not abuse its discretion in awarding punitive damages, where defendant represented a product as safe but "took no meaningful steps of any kind—even preliminary ones—to ensure the safety of its product"); *Borst v. Lower Manhattan Dev. Corp.*, 80 N.Y.S.3d 29, 31 (N.Y. App. Div. 2018) (workplace practices could create questions of fact as to "wilful and wanton disregard for the interests of others, justifying an award of punitive damages"). Summary judgment on punitive damages thus should be denied, but without prejudice to filing an appropriate Rule 50 motion at the close of plaintiffs' proof.

In contrast to its other recommendations, though, the Court agrees with defendants about Deborah's claim for loss of consortium. Defendants have argued for summary judgment on Deborah's claim for loss of consortium because James's alleged exposure to OT occurred in 1974, 12 years before James and Deborah married in 1986. Plaintiffs counter by citing to New York's "toxic tort statute of limitations," N.Y. CPLR 214-c. CPLR 214-c(2) states that New York's three-year limitations period for personal injury actions does not begin to run, in cases of latent effects of exposure, until the date on which injury is discovered or reasonably should have been discovered. CPLR 214-c, however, did not create any new causes of action, meaning that it did not affect how New York treats claims for loss of consortium:

> It is by now well settled that a cause of action for loss of consortium does not lie if the alleged tortious conduct and resultant injuries occurred prior to the marriage. Contrary to plaintiff's contention, the rationale underlying this rule is not limited to situations where the injuries to the spouse in question are manifest at the time of the marriage. As the Appellate Division explained below: "Consortium represents the marital partners' interest in the continuance of the marital relationship as it existed at its inception, not upon some guarantee that the marital partners are free of any preexisting latent injuries."
>
> Plaintiff's contention that the Legislature's enactment of the new discovery Statute of Limitations (CPLR 214–c) dictates a different result here is likewise unavailing. As we recently explained in *Enright v. Lilly & Co.*, 570 N.E.2d 198, *cert. denied*, 502 U.S. 868: "[CPLR 214–c] was directed at opening up traditional avenues of recovery by removing a procedural barrier that was unreasonable given the nature

> of DES injuries. Nothing in the legislation [however] suggests that the Legislature intended to expand the basis for liability" (*id.*, at 384). Similarly, we find plaintiff's reliance on the so-called revival statute (L.1986, ch. 682, § 4) to be misplaced. That provision merely temporarily revived certain previously time-barred claims—it did not act to create any new causes of action.

Anderson v. Eli Lilly & Co., 588 N.E.2d 66, 67–68 (N.Y. 1991) (citations omitted); *see also Rademacher v. Torbensen*, 13 N.Y.S.2d 124, 124 (N.Y. App. Div. 1939) ("If, at the time of his subsequent marriage, plaintiff's wife was disabled as a result of a previous negligent act by the defendant, the plaintiff took her as his wife in her then existing state of health and thus assumed any deprivation resulting from such disability."). Plaintiffs likely could have avoided this result if the claim for loss of consortium rested not only on pre-nuptial events but also some tort committed after their marriage. *See Torres v. Hyun Taik Cho*, 902 N.Y.S.2d 781, 783–84 (Sup. Ct. 2010) ("Contrary to defendants' claim, the actionable conduct here is not limited to the January 17, 1997 surgery. Rather, it includes a claim of malpractice based on Dr. Cho's failure to discover the foreign object and diagnose the related symptoms during the course of treatment following the first surgery, as well as malpractice by Dr. Cho and the St. Vincent's Medical Center based on the failure to detect the foreign object during the May 7, 2007 surgery. Those alleged departures occurred during the marriage, and Mr. Torres allegedly suffered injury during that period of time as a result of the malpractice."). James, however, has not alleged that defendants did anything to him after he married Deborah in 1986. "At its core, the *Rademacher* rule is a twist on the famous 'thin skull rule' of tort law: you take your spouse as you find him or her. When an unmarried person is tortiously injured, the liability created is limited to the damages recoverable by that person for personal injury. The courts have refused to allow third parties, by their voluntary act of marriage to the injured person, to in effect create an *ex post facto* liability for loss of consortium where none had existed before." *Walsh v. Armstrong World Indus., Inc.*, 700 F. Supp. 783, 786 (S.D.N.Y. 1988). Plaintiffs' sole case in support of their position, *Rothstein v.*

Tennessee Gas Pipeline Co., 661 N.E.2d 146, 148 (1995), does not change the above analysis. *Rothstein* does urge a generous reading of CPLR 214-c in general but did not even contain a claim for loss of consortium. *Rothstein* also does not cite *Anderson*; surely the New York Court of Appeals would have at least mentioned *Anderson* had it intended to fit that case under a general exhortation to read CPLR 214-c generously. Under these circumstances, the Court recommends summary judgment for defendants on Deborah's claim for loss of consortium.

IV. CONCLUSION

Suspicions about elevated bladder cancer risk from exposure to aromatic amines have been around since 1895. (Dkt. No. 56-28 at 2.) Since then, an extensive body of research literature has developed, and now, "[e]pidemiological studies have demonstrated a causal relationship between exposure to [OT] and urinary-bladder cancer in humans." (Dkt. No. 58-12 at 3.) The research literature never will include double-blind, randomized clinical trials, for obvious ethical reasons. Plaintiffs' experts have acknowledged that this limitation means that some uncertainty always will exist when assessing the causal relationship between OT and bladder cancer. Nonetheless, plaintiffs' experts have been careful not to oversell the conclusions in the research literature, and they have acknowledged the inferences required to form their opinions. So long as plaintiffs' experts continue to avoid overselling research results, their opinions are scientifically reliable and may be heard by a jury at trial. The jury also will hear cross-examination of plaintiffs' experts and direct testimony from defense experts; where the preponderance of the evidence falls regarding plaintiffs' negligence and other claims will be for the jurors to decide.

For all of the foregoing reasons, the Court respectfully recommends denying both of defendants' pending motions (Dkt. Nos. 56, 59) except to grant defendants summary judgment on Deborah's claim for loss of consortium.

V. OBJECTIONS

A copy of this Report and Recommendation will be sent to counsel for the parties by electronic filing on the date below. "Within 14 days after being served with a copy of the recommended disposition, a party may serve and file specific written objections to the proposed findings and recommendations." Fed. R. Civ. P. 72(b)(2); *see also* 28 U.S.C. § 636(b)(1). Any objections must be filed electronically with the Clerk of the Court through the CM/ECF system.

"As a rule, a party's failure to object to any purported error or omission in a magistrate judge's report waives further judicial review of the point." *Cephas v. Nash*, 328 F.3d 98, 107 (2d Cir. 2003) (citations omitted); *see also Mario v. P & C Food Markets, Inc.*, 313 F.3d 758, 766 (2d Cir. 2002) ("Where parties receive clear notice of the consequences, failure timely to object to a magistrate's report and recommendation operates as a waiver of further judicial review of the magistrate's decision.") (citation omitted). "We have adopted the rule that failure to object timely to a magistrate judge's report may operate as a waiver of any further judicial review of the decision, as long as the parties receive clear notice of the consequences of their failure to object. The rule is enforced under our supervisory powers and is a nonjurisdictional waiver provision whose violation we may excuse in the interest of justice." *United States v. Male Juvenile (95-CR-1074)*, 121 F.3d 34, 38–39 (2d Cir. 1997) (internal quotation marks and citations omitted).

"Where a party only raises general objections, a district court need only satisfy itself there is no clear error on the face of the record. Indeed, objections that are merely perfunctory responses argued in an attempt to engage the district court in a rehashing of the same arguments set forth in the original papers will not suffice to invoke de novo review. Such objections would reduce the magistrate's work to something akin to a meaningless dress rehearsal." *Owusu v. N.Y. State Ins.*, 655 F. Supp. 2d 308, 312–13 (S.D.N.Y. 2009) (internal quotation and editorial marks and citations omitted).

SO ORDERED.

/s Hugh B. Scott
Hon. Hugh B. Scott
United States Magistrate Judge

DATED: February 25, 2020